The Mechanism of Auricular Flutter and Auricular Fibrillation

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The renewed interest in the mechanisms underlying the various types of auricular arrhythmias stems primarily from the development of new methods of experimental approach. Electrical stimulation used by the majority of previous investigators to induce disturbances of auricular activation was known to effect auricular arrhythmias of only short duration, often restricted to the time of stimulation. Hence, the first fundamental step for further investigations was Scherf's publication in 1947 of a simple method to produce auricular arrhythmias of almost unlimited duration by application of aconitine to the exposed auricle of the dog. The method itself, and the first results of its use, developed into a serious challenge of apparently well established concepts almost universally accepted in the Anglo-American literature. The method, then, proved particularly useful in experiments performed with improved technics of exposure of both auricles and with highly developed technics of observation of the disturbed auricular function, as introduced by Prinzmetal. However, while this new experimental approach seemed to weaken the fundamental concept derived from the basic experiments of Lewis, new evidence in its support was provided by the work of Rosenblueth and his group. These and other controversial data of the past were a great stimulus toward reopening the discussion.

The Principal Theories of Continuous Rapid Activity of the Auricles

In discussing this problem the first step is to present the theories which have been advanced for these rhythms. The principal ones are:

I. The theory of multiple foci with rapid discharge, first advanced by Engelman, was favored originally by the Viennese school and utilized as an explanation of fibrillation up to the present by Kisch.

II. The theory of a single ectopic focus with rapid discharge was advocated by Rothberger and Winterberg and by Scherf as a result of his first experiments with aconitine and accepted by Prinzmetal.

III. The theory of a single circulating wave was conceived by Lewis, and in a modified form by De Boer. A related concept has been advanced by Ashman and Barker and co-workers to explain the mechanism of supraventricular tachycardias other than auricular flutter and fibrillation.

IV. The concept of multiple and minute re-entries of the wave of excitation, was proposed by Garrey and also applied by Wiggers in his studies on ventricular fibrillation.

V. The concept of multiple self sustained microsystole with "extreme reduction of consumption of systolic energy" was advanced by Wenckebach and Winterberg.

The numerous arguments which have been brought forward in favor of and against each of the above concepts have many aspects in common. Their essential points can be summarized in connection with the following three salient questions:

1. Has re-entry and circus movement been proved in the heart of experimental animals and of man? Can such a mechanism alone account for the development of auricular flutter and fibrillation?

Re-entry and continued slow circulation of the cardiac impulse has been demonstrated beyond question by Mines and Garrey in excised parts of the auricles and ventricles of experimental animals. Incomplete evidence of its rapid form was provided by Lewis and co-workers in their attempts to trace the path-
way of the wave of excitation in experimental auricular flutter. Objections to the latter were raised very soon by Rothberger who pointed out the lack of continuous muscular structures within the narrow pathway as supposed by Lewis. However, the argument that Lewis was unable to trace the wave of excitation over its entire pathway around the venous ostia became invalid when unquestionably complete circuits of the impulse in the auricles around artificial obstacles of sufficient perimeter and completely surrounded by conductive tissue were demonstrated by Rosenblueth and co-workers. Further and important arguments against a single mother wave initiating auricular fibrillation and flutter can be considered to be the experiments of Brams and Katz who observed the persistence of both types of arrhythmia in both parts of the chamber following complete separation of the auricle into two parts, and similar observations of Prinzmetal following scarification of the region of the supposed pathway of Lewis' mother wave.

Lewis also believed that evidence for a circus movement in human auricular flutter could be derived from the rotation of the electrical axis of auricular activation constructed from three-dimensional chest leads. His calculations performed in only a few patients with auricular flutter were later confirmed by others with a larger amount of material and recently amplified with the help of similar analysis of simultaneous esophageal and intracardiac leads in patients with auricular flutter and fibrillation. However, as far back as 1923, Rothberger had stressed the weakness of the argument by pointing out that the auricular complex (F wave) in auricular flutter necessarily represents the activation of the entire mass of both auricles taking place in all directions, rather than the reflection of electrical phenomena associated with the activation of a narrow muscle band around the venae cavae, as supposed by Lewis. Prinzmetal, after repeating Lewis' calculations, came to the conclusion that the diphasic character of auricular complexes in flutter is a manifestation of both auricular depolarization and repolarization occurring in rapid alternation. Such a concept, invalidating one of Lewis' principle arguments, seems convincing particularly in view of similar events taking place with the onset and progressive acceleration of ventricular tachycardias, resulting eventually in the typical see-saw appearance of the electrocardiogram. The same author also failed to observe in high-speed-camera motion pictures, even under high magnification, any type of motion which would suggest a circulating movement in the fibrillating auricles of dogs and of man.

While, thus, no direct evidence is at hand which would prove the existence of a circulating type of movement in human auricular flutter and fibrillation, proof for re-entry of the cardiac impulse in other than auricular tissue is available. A considerable number of unquestionable clinical instances of reciprocal beating has been reported. Recently repetitive and multiple re-entry of the cardiac impulse within the A-V node was strongly suggested on the basis of a detailed analysis of electrocardiograms in clinical instances exhibiting complex, rare and otherwise unexplained arrhythmias.

2. Is there sufficient evidence for the presence of one or more rapidly firing foci which would account satisfactorily for the development of auricular flutter and fibrillation in the experimental animal and in man?

Undoubtedly, the strongest point of evidence in this respect is the fact that all three types of rapid auricular arrhythmias—tachysystole, flutter, and fibrillation—and transitions of one into the other, can be produced experimentally by focal application of an electrical or pharmacologic stimulus to the auricle. Further and even stronger support is provided by the fact that at least tachysystole and flutter can be acutely stopped by cooling the initiating focus or separating it from the rest of the auricle, and by the prompt reappearance of rapid auricular activity by rewarming the area of the focus. While all these observations appear, at first glance, to be incompatible with the circus movement theory, consideration of additional experimental data and of clinical experience is apt to prevent unconditional acceptance of this mechanism as the sole responsible factor for the development of all types of rapid auricular activity as proposed
by Prinzmetal in the form of his concept of the "unitary nature of auricular arrhythmias." Very early in the course of such an investigation, it was pointed out by Garrey that following division of the fibrillating auricles into two and even four equal parts, each of them persists in its uncoordinated and rapid activity. Experiments on fibrillating and fluttering auricles and ventricles, with identical results, were reported later by Brams and Katz. Furthermore, it has been repeatedly shown that experimental auricular or ventricular fibrillation can be induced by a single induction shock applied in a certain "vulnerable" time period at the beginning of the repolarization of the respective chamber, and this mechanism can also be terminated by a similar procedure. Such facts are hard to reconcile with the concept of a pure "unifocal" theory of the mechanism of this disturbance of rhythm.

Further objections against any "focal" origin of auricular arrhythmias stem from pharmacologic and clinical experience rather than from direct evidence. This refers mainly to the different response of simple auricular tachysystole on one hand, and auricular flutter and fibrillation on the other hand, to (1) vagal stimulation, (2) treatment with digitalis, (3) their abrupt onset and termination and (4) their failure to recur clinically after interruption. Also, the persistence of one or more foci of rapid impulse formation over many years and the necessarily associated shortening of the refractory phase over long periods of time are inconsistent with known physiologic facts. Such considerations forced some authors to invoke mechanism of a circus movement not only for flutter and fibrillation but also for other types of supraventricular tachycardias.

3. Is there reason for, and the possibility of, reconciling the two divergent standpoints?

As outlined in the preceding paragraphs none of the proposed theories—single or multiple circus movements, single or multiple rapidly discharging foci—can by itself apparently ac-count for all the diverse phenomena associated with the appearance, perpetuation and termination of auricular flutter and fibrillation produced experimentally or observed in clinical instances. Therefore, the logical question arises whether a compromise standpoint could contribute to the solution of the unsolved problem. Attempts in this direction are not wanting in the writings of past and present investigators. Wenckebach and Winterberg have pointed out in their monograph that once a point of re-entry has developed in excitable tissue it can be considered as a focus firing rapid impulses in all directions. Rothberger in his discussion of the genesis of ventricular fibrillation visualized the chain of events as follows: An initial single focus discharging impulses at a high and regular rate effects the surrounding myocardium progressive shortening of the refractory period, and at the same time a progressive decline of conductivity; thus conditions are created favorable for the development of local re-entries of the impulses. While this is at first restricted to an area around the point of impulse formation, the same process could suddenly extend over the whole affected chamber with the result of innumerable minute re-entries, each of them discharging impulses in all directions and terminating in a condition of more or less uncoordinated activity, termed fibrillation. A similar mechanism was suggested by Scherf to be operating in auricular arrhythmias. It is perhaps of interest that Scherf, originally a decided proponent of a unifocal theory of the mechanism of all three types of auricular tachycardias, concluded as a result of later experiments that a rhythmic stimulus may be acting in auricular tachycardias and a continuous stimulus in auricular flutter and fibrillation; he further concluded that fibrillation cannot be due to a single mechanism. According to his more recent view, Scherf believes a circus movement is not the cause but a concomitant feature of rapid auricular activity.

**Personal Opinion**

This opportunity to resurvey the literature has not materially altered the viewpoints expressed previously, after a similar survey in
Auricular flutter and fibrillation is initiated by a single impulse coming in a vulnerable period of the heart cycle. This may be a sinus impulse when the heart’s function is markedly depressed, or a premature ectopic one when the heart is less depressed. Thus, the degree of prematurity of the impulse sufficient for perpetuation of the disturbance of rhythm will vary more or less inversely with the impairment of the heart’s functions particularly with the prolongation of the refractory phase. Of course, a continued acceleration of the sinus pacemaker, or of a rapid ectopic one, may help to sustain auricular flutter and fibrillation once it is initiated. Hence, in certain instances, depression or abolition of an ectopic focus will permit auricular flutter or fibrillation, sustained by its activity, to disappear. In addition, the borderline between a rapid tachysystole with radial spread of the impulse and a rapid auricular activity giving rise to the peculiar phenomena of flutter is a variable one depending on the functional state of the auricle at the time. In other words, not every tachycardia of the auricle at a rate of 300 or more is flutter (or fibrillation) even though the waves expressing auricular activity occupy the entire auricular cycle. As has been mentioned this may merely mean that both auricular depolarization and repolarization are electrically manifest.

It would seem that the perpetuation of auricular flutter and fibrillation depends on the development of multiple re-entries. In flutter, the cyclic repetition of these re-entries is practically fixed in pattern. In fibrillation, no fixed pattern for the repetitive re-entries exists and in impure flutter there is some tendency to a fixed pattern, but this is imperfect. By use of such a concept the beautiful studies of Lewis and his associates, referred to earlier, can be applied, not as Lewis did in oversimplifying the subject to a mother ring, but rather to the multiple simultaneous circulating waves, each with its “gap” and its “head” trying to reach and swallow its “tail.” In this fashion, the arguments of the “circuit movement exponents” can be accomodated without the need of locating a primary circulating wave.

The conditions for re-entry are ripe in the auricular syncitium provided only that an impulse can come early enough in the refractory phase so that it reaches a point of branching while one arm is still refractory and prevents its passage, and the other arm is not. Under these conditions the impulse will travel through a loop, short or long, and return to the point of branching in a retrograde fashion. If, now, this branching is no longer refractory the impulse will re-enter the myocardium. Since the path is usually short, the re-entering impulse will be very premature and this, in turn, will enable it to set up re-entries in neighboring regions, and so re-entries will pile up, spread over the auricles, and perpetuate themselves. The ease of setting up re-entries is obviously facilitated in the case of diseased auricles for several reasons. First, the refractory period is prolonged by disease making it possible more often for the impulse to reach points of branching in which one limb is refractory and the other is not. Second, the effect of disease is to make the functional state of neighboring parts of the syncitium more unequal so that the frequency with which the impulse encounters loops ripe for re-entry is enhanced. Third, the sort of state established experimentally by Rosenblueth and his co-workers is more apt to occur; such states can be due to anatomic changes or to functional ones following upon ischemia, hypoxia or altered states of the innervation, or of electrolyte balance.

It would follow from the above that the more diseased the auricles are, the less premature need be the impulse initiating and/or sustaining the mechanism of multiple re-entries responsible for flutter and fibrillation, the greater also the ease of the re-entries perpetuating new re-entries, and the smaller the chance to overcome the mechanism pharmacologically. The saving grace for the last, however, is the fact that improving the physiologic state of the heart handicaps the facility of re-entries to continue. To this can be added the fact that drugs which “close the gap,” as established by the school of Lewis, also offer a positive approach to the handling of these important clinical entities.

It must be stated in conclusion that this personal opinion is merely the working hypothesis of the authors and in no sense an
established fact. Time alone will offer the ultimate solution, but we predict that auricular flutter and fibrillation will turn out to be more complex than is suggested by any unitary theory. Moreover, it should be kept in mind that the experiments used in evidence are performed on animals for the most part with normal hearts whereas clinical auricular flutter and fibrillation develops in patients with heart disease. So far, nobody has been able to reproduce in animals conditions which would approximate all anatomic and functional alterations present in diseased human hearts with auricular flutter and fibrillation. Thus, experimentally produced disturbances of cardiac rhythm, and the mode of their production, may not be identical with seemingly similar events occurring under certain pathologic conditions in man. Structural and/or biochemical factors, not known or so far not suspected, may precipitate, or facilitate, the development of various types of auricular arrhythmias in man. There remains for the future this matter of correlation of further experimental and clinical investigations.

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